CASE REPORT

Isolated right ventricular infarction: a diagnostic challenge

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SUMMARY

A 73-year-old woman was admitted to the emergency room due to sudden-onset dyspnoea, altered mental status and haemodynamic instability. ECG showed a junctional rhythm, T-wave inversion in I, aVL and V2-V6 (present in a previous ECG), and no ST/T changes in the right precordial leads. Transthoracic echocardiography, however, revealed a severe depression of global systolic function of right ventricle with akinesia of free wall and a normal left ventricular function. Coronary angiography showed an occlusion of the proximal segment of the right coronary artery, which was treated with balloon angioplasty, and a chronic lesion of the anterior descending artery. The patient had a good recovery and was discharged on the 14th day. Myocardial perfusion scintigraphy (stress and rest) was performed a month later, showing a fixed perfusion defect in the apex and anterior wall (medium-apical), with no signs of ischaemia.

BACKGROUND

Isolated right ventricular (RV) infarction is an extremely rare condition and its diagnosis may be challenging. Although RV infarction may occur in association with infarction of the inferior wall of the left ventricle (LV),[1] only a proportion of these cases presents with haemodynamic compromise.[2 3] RV involvement always entails a poorer outcome, as cardiogenic shock, severe bradycardia and in-hospital mortality are much more common in this context.[4 5]

As in this case, isolated RV infarction may present similarly to pulmonary embolism and can share identical echocardiography findings. It should be suspected in patients with unexplained shock and negative CT pulmonary angiogram. The management of RV infarction involves a range of specific steps including coronary artery angiography and revascularisation.

CASE PRESENTATION

We present a case of a 73-year-old woman admitted to the emergency room due to sudden-onset dyspnoea, prostration and slowed speech. The beginning of dyspnoea had occurred 4 h earlier and was accompanied by diaphoresis and malaise. At admission, the patient was hypotensive (70/30 mm Hg) and bradycardic, with signs of poor peripheral perfusion. Her body temperature was normal, she had a slightly increased respiratory rate and saturation of peripheral oxygen was 92%. Jugular distention was visible but there was no paradoxical pulse. No changes in heart sounds and no lung auscultation were noted. There was neither symmetrical nor asymmetrical oedema of lower extremities. Despite being prostrate, the patient remained oriented and answered questions with slowed speech (Glasgow Coma Scale (GCS)=13).

Her medical history included hypertension and an ischaemic stroke 8 years prior, with consequential dysarthria and chronic use of carvedilol 6.25 mg two times a day, telmisartan 40 mg four times a day, amlodipine 5 mg four times a day, furosemide 40 mg four times a day and clonidine 0.15 mg four times a day.

INVESTIGATIONS

ECG showed junctional rhythm, heart rate of 35 bpm, T-wave inversion in I, aVL and V2-V6 (present in a previous ECG), and no ST/T changes in the right precordial leads (figure 1A, B). Angio-CT excluded pulmonary embolism and aortic dissection. Transthoracic echocardiography showed normal global and segmental systolic function of LV, severe depression of the global systolic function of RV (tricuspid annular plane systolic excursion (TAPSE)=4 mm) with akinesia of RV free wall and evidence of neither mechanical complications nor pericardial effusion (figure 2A–E). Blood tests also became available, showing a serum creatinine of 1.8 mg/dL and normal values of cardiac markers. In the meanwhile, an episode of monomorphic ventricular tachycardia was noted. An urgent coronary angiography was performed, which revealed a balanced circulation and occlusion of the proximal segment of the right coronary (RC) artery (figure 3C, arrow), and a chronic lesion of the proximal segment of the anterior descending artery (figure 3A, B).

TREATMENT

Initial treatment in the emergency room included intensive intravenous fluid administration and volume expansion, dopamine perfusion and placement of a temporary pacemaker through femoral vein. After angiography, a balloon angioplasty of the proximal segment of the RC was performed (figure 3D–F). An intra-aortic balloon was placed, dopamine was replaced by dobutamine, and abciximab and unfractionated heparin were administered. The patient was admitted to the intensive care coronary unit.

OUTCOME AND FOLLOW-UP

There was progressive haemodynamic improvement, especially after restoration of sinus rhythm (day 3), which allowed the withdrawal of inotropic support, pacemaker and intra-aortic balloon. Peak troponin I

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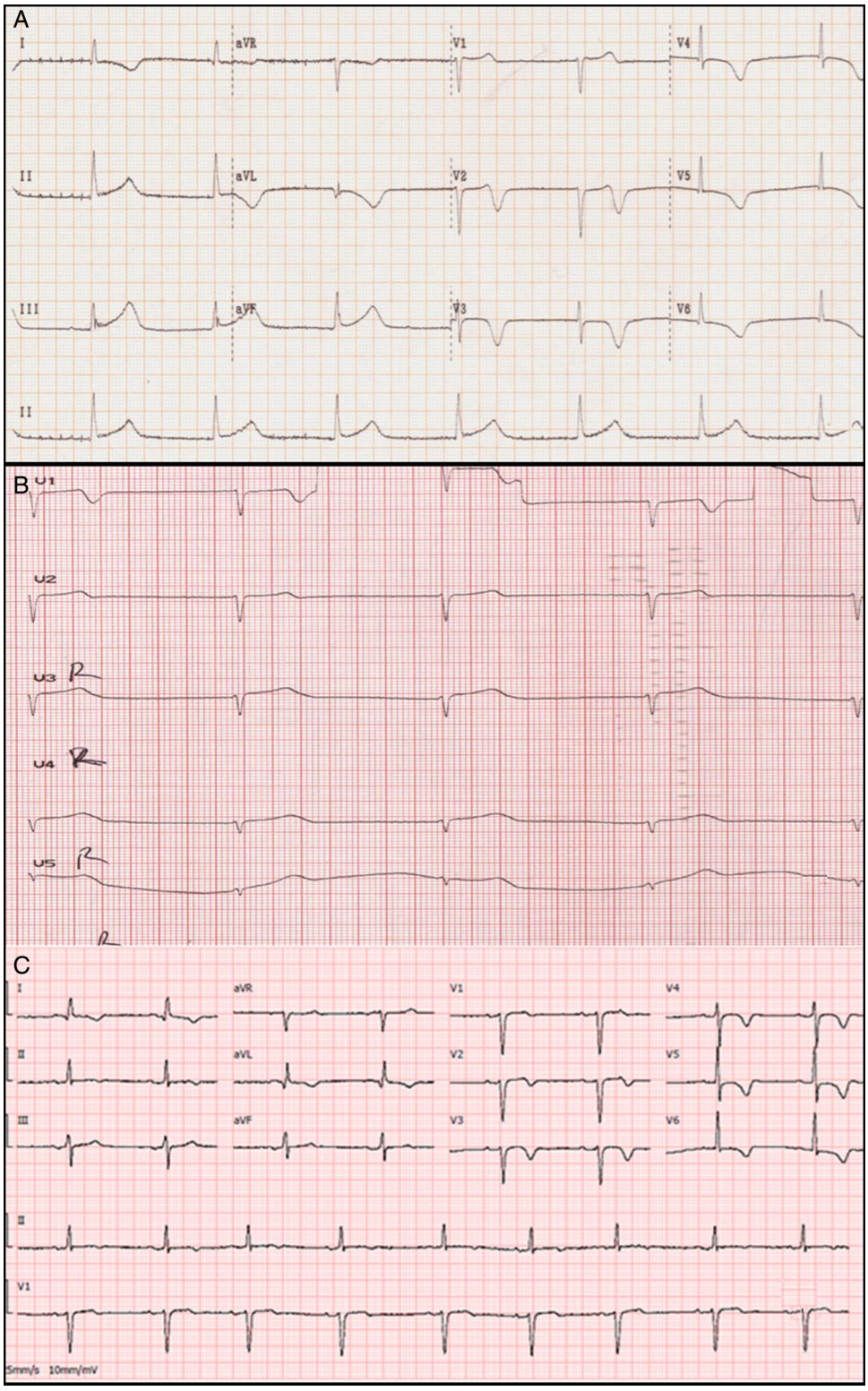

Figure 1 Twelve-lead ECG (A) and right precordial leads (B) showing bradycardia (junctional rhythm, heart rate of 35/min), T-wave inversion in I, aVL and V2-V6 (present in a previous ECG), and no ST/T changes in the right precordial leads (V3R-V6R). Six months after the event, 12-lead ECG had no new ST–T changes (C).

was 4.8 ng/mL, creatine kinase MB (CK-MB) was 178 U/L and CK-MB mass was 149 ng/mL. Haemoglobin remained stable and serum creatinine level dropped to 1.3 mg/dL. The patient was discharged on the 14th day.

A month later, she presented for myocardial perfusion scintigraphy (stress and rest), to evaluate ischaemia in the territory of the anterior descending artery. The scintigram showed a fixed perfusion defect in the apex and anterior wall (medium-apical), and no evidence of ischaemia (figure 4). Six months later, the patient remained asymptomatic, her ECG had no new ST/T changes (figure 1C), and transthoracic echocardiography showed recovery of global and segmental function of right ventricle—TAPSE=17 mm; tissue Doppler systolic wave of lateral tricuspid annulus=12.5 cm/s (figure 2F–J).

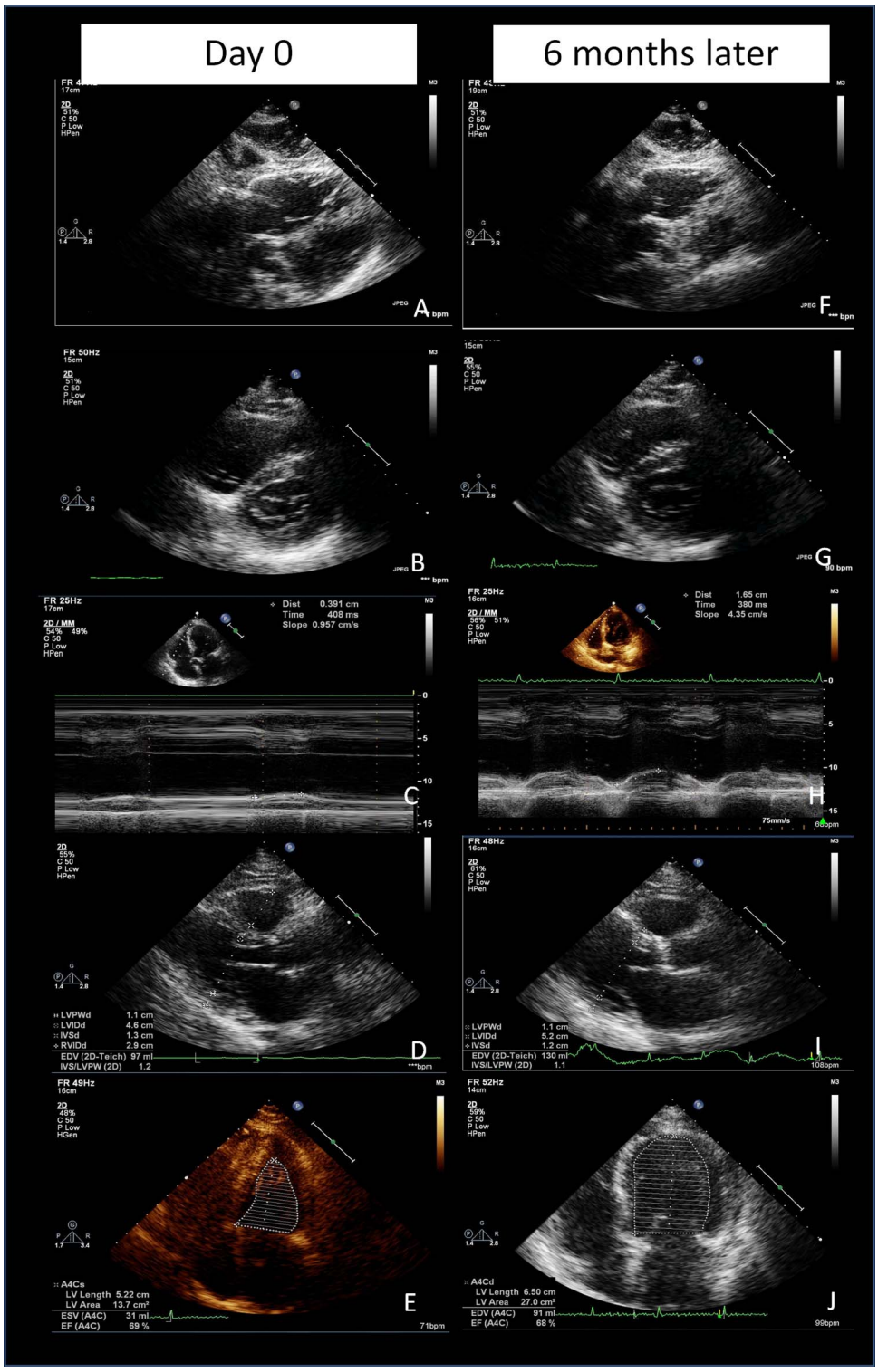

Figure 2 Two-dimensional transthoracic echocardiogram at admission (A–E) and 6 months later (F–J), showing recovery of right ventricle dilation and function, and normal global and segmental function of left ventricle.

DISCUSSION

Clinical presentation of RV infarction may be very distinct. The classic triad consists of hypotension, clear lung fields and raised jugular venous pressure, but other signs such as hepatomegaly, high-grade atrioventricular block, tricuspid regurgitation, cardiogenic shock and cardiac tamponade may also point in that direction. A sign of the presence of haemodynamically significant RV infarction is an exaggerated response to preload reducing agents such as nitrates, morphine or diuretics.[6]

Our case was a diagnostic challenge for several reasons: the patient did not refer thoracic pain, anamnesis was limited due to the patient's altered mental status and ECG had no new ST–T changes. Also, the patient presented with bradycardia, which could explain her clinical status. Non-ischaemic causes of sudden-onset dyspnoea such as pulmonary embolism, cardiac tamponade, pneumothorax, acute pulmonary oedema, airway obstruction and pneumonia with atelectasis, were then excluded by physical examination and angio-CT. However, in a patient in

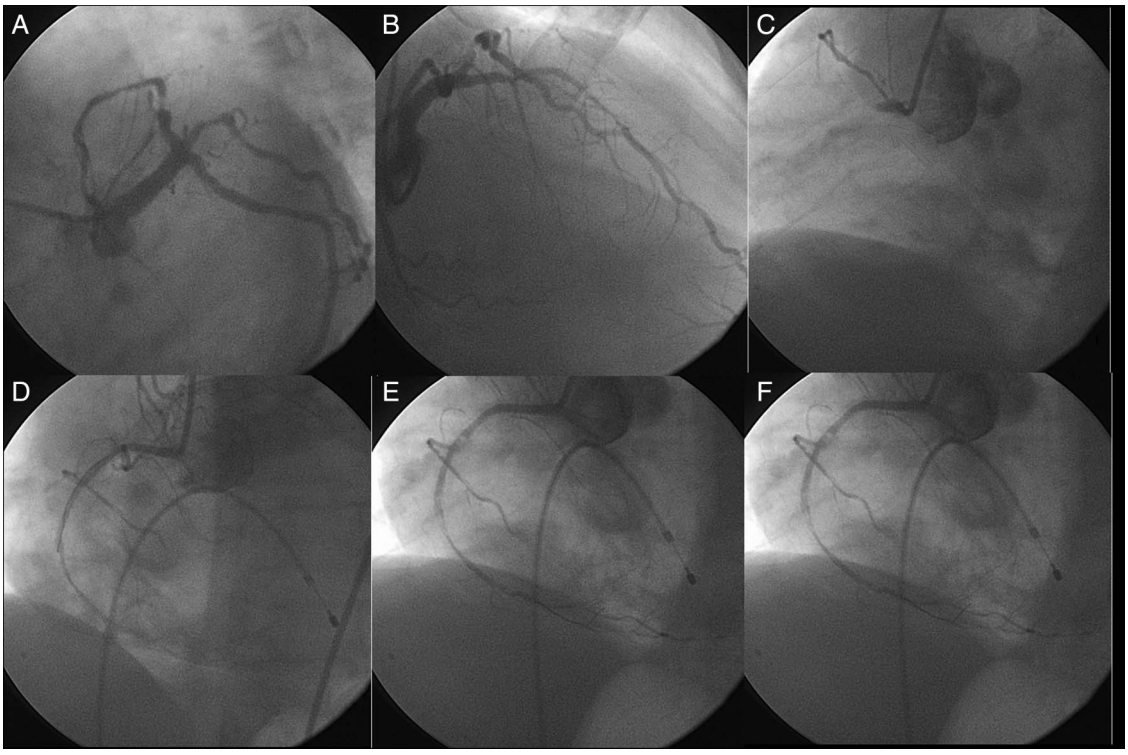

Figure 3 Coronary angiography showing a chronic lesion of the proximal segment of the anterior descending artery (A and B) and an acute occlusion of the proximal segment of the right coronary artery (C, arrow) treated with angioplasty and stenting (D–F).

shock, with cardiovascular risk factors, previous cardiovascular events and basal ST–T changes, it is necessary to exclude coronary artery disease, and echocardiography may be crucial.[7] In fact, transthoracic Doppler echocardiography has a vital role in the diagnosis of isolated RV infarction, assessment of LV ischaemia and exclusion of other diagnoses.[8] The most frequent echocardiographic findings are RV dilation and dysfunction (global and segmental), low pulmonary arterial pressure and dilated hepatic veins. Tricuspid regurgitation, paradoxical movement of interventricular septum and a right-to-left interatrial shunt may also be present due to increased right chamber diastolic pressures. Echocardiography may also add prognostic data, as long as the presence of RV dysfunction on early echocardiography denotes a poor outcome, particularly when the RV dysfunction is associated with LV dysfunction.[9]

The treatment of RV infarction also has some special features. The key concept is that acute RV shock has the same high mortality as LV shock, and the treatment should be prompt and as specific as possible. As in patients with LV cardiogenic shock due to ischaemia, reperfusion offers the best chance of survival and complete recovery.[10] Besides, in the specific case of RV ischaemia/infarction, it seems that even late reperfusion, >12 h after the onset of symptoms, may be beneficial for prognosis and functional recovery. Nowadays, reperfusion is established mainly by percutaneous coronary intervention, but thrombolysis remains an option when the former is not readily available.

Other treatment is the maintenance of adequate RV filling pressures, mainly by fluid volume administration. However, this therapy must be performed with caution—if carried out in excess, it may worsen the shifting of the interventricular septum to the left with consequent limitation of LV filling and a decline in cardiac output. As diuretics and vasodilators reduce RV filling, they should be administered with close monitoring or not used at all.

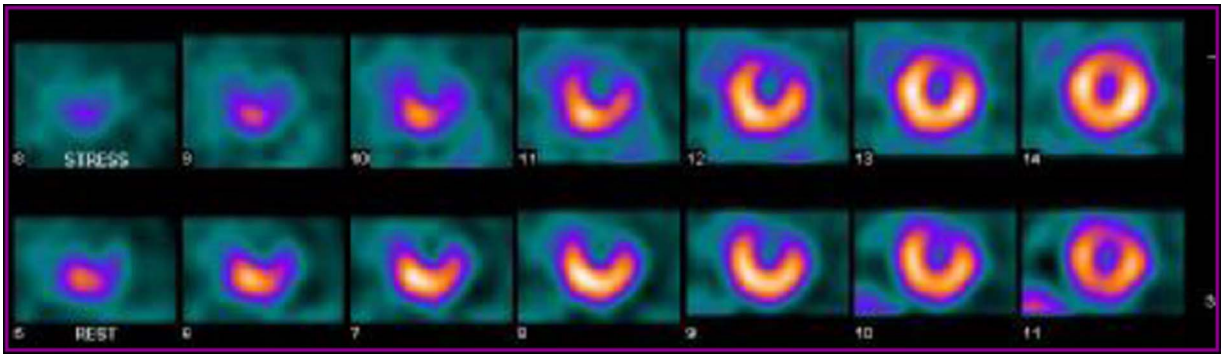

Figure 4 Myocardial perfusion scintigraphy (stress and rest) performed a month after the event, showing a fixed perfusion defect in the apex and anterior wall (medium-apical) and no evidence of ischaemia.

If these measures are insufficient, inotropic therapy should be started. Although very few data are available in the literature, the use of an intra-aortic balloon pump in isolated RV infarction may be beneficial even in the presence of intact LV contractility.[11] The mechanisms involved are speculated on, but seem to be the improvement of coronary perfusion and of LV haemodynamics, which is impaired by RV dysfunction. Some studies have shown a positive effect of nitroprusside infusion for afterload reduction; infusion may be needed when there is concomitant left ventricular dysfunction. Inhaled nitric oxide has also been associated with rapid haemodynamic improvement, especially when combined with dobutamine.[12 13] In the presence of new onset atrial fibrillation or atrioventricular block in association with bradycardia, the preferred treatment is atrioventricular sequential pacing.[14]

Learning points

- The difficulty in diagnosing an isolated right ventricular (RV) infarction and the specificities in its management account for a high in-hospital mortality rate.
- Isolated RV infarction has a variable presentation but the classical triad consists of hypotension, clear lung fields and raised jugular venous pressure.
- Differential diagnosis should include other acute conditions such as pulmonary embolism, pneumothorax, cardiac tamponade, and pericardial and valvular disease.
- Transthoracic echocardiography may be of extreme value in the assessment of shock patients or in the suspicion of isolated RV infarction and should be performed as soon as possible.
- RV infarction management should focus on restoring RV filling pressure, inotropism and sinus rhythm as well as revascularisation.

Competing interests None declared.

Patient consent Obtained.

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REFERENCES

1 Bates ER. Revisiting reperfusion therapy in inferior myocardial infarction. *J Am Coll Cardiol* 1997;30:334–42.
2 Lisbona R, Sniderman A, Derbekyan V, *et al*. Phase and amplitude imaging in the diagnosis of acute right ventricular damage in inferior infarction. *Clin Nucl Med* 1983;8:517–20.
3 Martin W, Tweddel A, McGhie I, *et al*. The evaluation of right ventricular function in acute myocardial infarction by xenon-133. *Nucl Med Commun* 1989;10:35–43.
4 Berger PB, Ryan TJ. Inferior myocardial infarction: high-risk subgroups. *Circulation* 1990;81:401–11.
5 Mehta SR, Eikelboom JW, Natarajan MK, *et al*. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll Cardiol* 2001;37:37–43.
6 Mittal SR. Isolated right ventricular infarction. *Int J Cardiol* 1994;46:53–60.
7 Sia YT, O'Meara E, Ducharme A. Role of echocardiography in acute myocardial infarction. *Curr Heart Fail Rep* 2008;5:189–96.
8 Singhal AM, Ilangovan S, Mehta S, *et al*. Isolated right ventricular infarction followed by posterior left ventricular infarction after a few days. *Acta Cardiol* 1984;39:307–12.
9 Engström AE, Vis MM, Bouma BJ, *et al*. Right ventricular dysfunction is an independent predictor for mortality in ST-elevation myocardial infarction patients presenting with cardiogenic shock on admission. *Eur J Heart Fail* 2010;12:276–82.
10 Jacobs AK, Leopold JA, Bates E, *et al*. Cardiogenic shock caused by right ventricular infarction: a report from the SHOCK registry. *J Am Coll Cardiol* 2003;41:1273–9.
11 McNamara MW, Dixon SR, Golstein JA. Impact of intra-aortic balloon pumping on hypotension and outcomes in acute right ventricular infarction. *Coron Artery Dis* 2014;25:602–7.
12 Inglessis I, Shin JT, Lepore JJ, *et al*. Hemodynamic effects of inhaled nitric oxide in right ventricular myocardial infarction and cardiogenic shock. *J Am Coll Cardiol* 2004;44:793–8.
13 Lahm T, McCaslin CA, Wozniak TC, *et al*. Medical and surgical treatment of acute right ventricular failure. *J Am Coll Cardiol* 2010;56:1435–46.
14 Tobinick E, Schelbert HR, Henning H, *et al*. Right ventricular ejection fraction in patients with acute anterior and inferior myocardial infarction assessed by radionuclide angiography. *Circulation* 1978;57:1078–84.